CLINICAL PROCEEDINGS

of the

CHILDREN'S HOSPITAL

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CLINICAL PROCEEDINGS

OF THE CHILDREN'S HOSPITAL

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PROTHROMBIN STUDIES IN PEDIATRICS

Jean Lockhart, M.D.

With the discovery of vitamin K and its relationship to prothrombin, renewed interest has centered about the process of clotting. On the whole, pediatrics has shared in the benefits of the resulting research, although in few instances were children made the subjects of hematologic studies. This is particularly true of newborns, whose total circulating blood volume is so limited. Yet it was known even in biblical times that newborn babies may bleed, and the covenant specified that "he that is eight days old shall be circumsized among you" (Genesis XVII:12). It is now clear that the danger of so-called hemorrhagic disease of the newborn has passed by the eighth day, and that the prothrombin time, as measured by the one-stage method, starts to fall shortly after birth, and is usually normal by the fifth day. The relationship between these two facts, however, is not entirely clear.

In 1940, Quick studied the prothrombin concentration of the blood of 30 healthy newborns, from birth to eight days[1]. His results, while they tend to show the restoration to normal of the prothrombin in five days, reveal a surprising scatter in results. They are duplicated in Figure I.

Many comparable studies have been made to test clotting factors in the blood of newborns (including prothrombin studies using the two-stage method of Brinkhaus, Smith and Warner), but always on a relatively limited number of babies, and usually with rather scattered results. The difficulties are apparently inherent in the problem: to draw 4.5–6 cc. of *unclotted* blood from the vein of a newborn infant.

The advantages that a prothrombin test on *capillary* blood would possess are manifest. A number of such tests have been devised. Quick himself describes a micromethod, "for clinical purposes" of prothrombin determination on young infants:

"a drop of blood obtained by a heel or ear lobe puncture is put on a glass slide, and mixed with a drop of equal size of thromboplastin. The mixture is slowly stirred with a fine pointed stirring rod. By holding the glass slide over a light, the exact clotting time can readily be determined. Normal blood will clot in 15–20 seconds[2]."

A more elaborate test was devised by Abramson and Weinstein[3], using blood from a heel puncture. The following were to be mixed rapidly in a clean hanging drop slide: 10 c.mm. M/40 calcium chloride, 10 c.mm. thromboplastin, and 10 c.mm. whole, unoxalated blood. This test was based on the observation that there is essentially no difference between

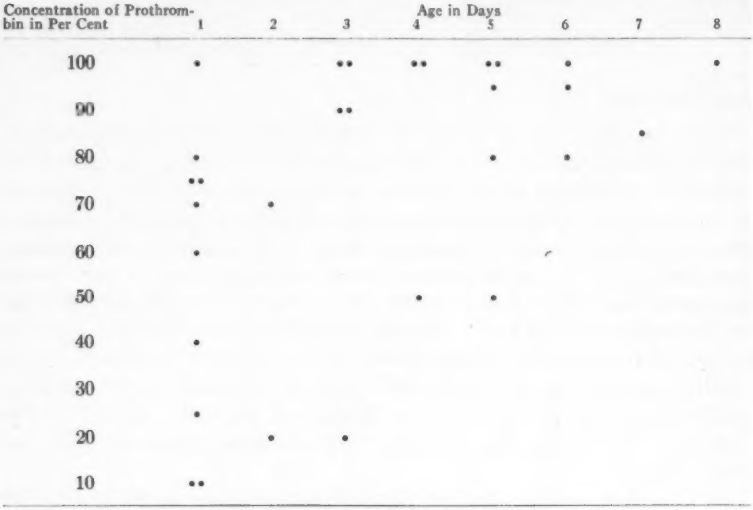

FIGURE I

the clotting time of recalcified oxalated plasma and unoxalated plasma or blood, provided an excess of thromboplastin is added.

Hoffman and Custer perfected a micromethod for determining prothrombin time on fresh capillary blood, the emphasis being on standard physical conditions[4]. Their procedure involved heating a glass slide to 37.5°C., placing 0.05 ml. thromboplastin on the slide, adding 0.05 ml. whole blood obtained from heelprick, and timing the clot formation, while stirring the mixture with a rake which is stroked at a set time interval. Accuracy of the test depends upon adherence to these rigid circumstances.

In an effort to determine the practicality and accuracy of a capillary prothrombin test, colored newborns on the staff service of Garfield Hospital were subjected to prothrombin tests during November and December, 1953, as follows: Daily prothrombin determinations were made on each baby by two methods simultaneously, the Quick method, using venous blood which was tested routinely by the Garfield Hospital laboratory, and a capillary method which will be described. Twenty such determinations were made, the babies and their mothers having received no supplementary vitamin K.

The capillary test used was outlined by Dr. Benjamin Manchester at a recent exhibit at the District of Columbia Medical Society convention. The following equipment is needed: a Kline slide, Sahli pipettes (graduated to

20 c.mm.), a stopwatch, and thromboplastin from Difco Laboratories ("Bacto Thromboplastin"). The thromboplastin is refrigerated until used, and then each ampule is made up so that 4 cc. normal saline is added to it, and the suspension is incubated for 15–20 min. at 40°C. It is then quick-frozen until use at the bedside. There, the slide is washed with hot water, and the thromboplastin is warmed in running hot water for 5–8 minutes, then both slide and thromboplastin are rested on paper or a towel (for insulation). 20 c.mm. of the thromboplastin is pipetted into one of the slide depressions. 20 c.mm. fresh blood from a deep heelprick is added to this, and timing is started immediately, while the slide is gently rolled. A control is tested at the same time, under the same conditions, and a given lot of thromboplastin is used until the control is 14–21 seconds, then discarded.

It will be noted that this test combines the simplicity of Quick's original capillary method with the more rigid physical condition standards of Hoffman and Curtis. Results of the tests are shown in Figures II and III.

It is apparent from these results that neither the venous nor the capillary

No.	Date	Patient	Age (days)	Venous Method					Capillary Method		
				Control		Baby			Control	Baby	
				Whole plasma	Dil plasma	Whole plasma	Dil plasma	% Activity	Sec	Sec	% Activity
1	11/3	Girl L.	2	13	28	13	35	100	—	—	—
2	11/3	Boy S.	4	13	28	15	75*	70	—	—	—
3	11/4	Girl L.	3	—	—	—	—	—	20	35	57
4	11/4	Boy F.	1	13	27	14	34	73	20	22	90
5	11/5	Boy F.	2	13	27	19	46	30	22	35	63
6	11/5	Girl T.	1	13	27	19	50	30	22	22	100
7	11/6	Girl T.	2	13	27	18	50	40	18	45	40
8	11/10	Girl D.	1	13	27	14	34	80	15	15	100
9	11/12	Girl H.	4	13	27	14	33	80	25	38	66
10	11/12	Girl D.	3	13	27	16	50	60	25	42	60
11	11/13	Boy B.	1	13	27	14	40	80	23	23	100
12	11/13	Boy W.	2	13	27	18	45	45	23	29	79
13	11/16	Boy B.	4	13	27	13	42	80	24	30	80
14	11/16	Girl E.	4	13	27	15	39	70	24	27	89
15	11/18	Boy D.	1	13	27	20	70	29	23	35	66
16	11/19	Boy D.	2	13	27	19	80	30	23	26	89
17	11/25	Girl B.	3	14	28	14	28	100	26	46.5	56
18	11/30	Girl O.	2	13	27	18	36	38	23	30	77
19	11/30	Boy C.	2	13	27	15	32	64	23	23	100
20	12/2	Girl T.	1	13	27	15	50	70	28	38	74
	11/18	Control		13	27	13	30	100			

* Hemolyzed.

FIGURE II

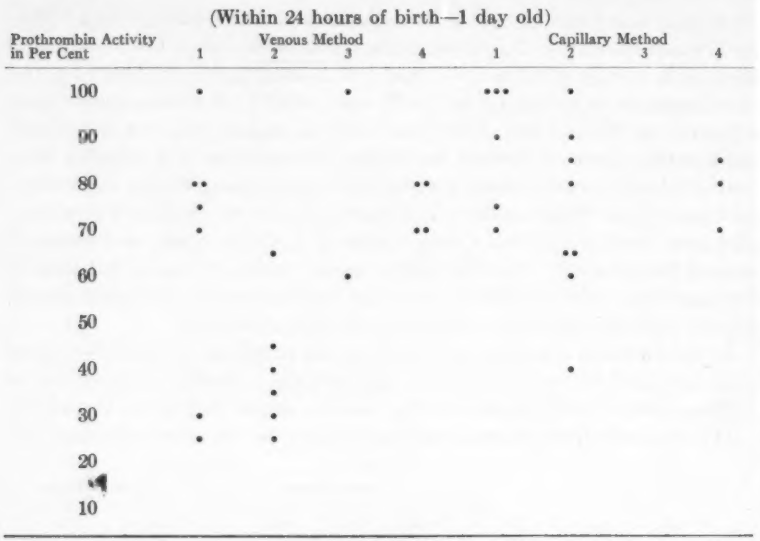

FIGURE III

prothrombin time determinations are as consistent as could be hoped, either with expected values in newborns, or with day by day results. Nor is there much correlation between the two methods, for example, one day old Baby Boy D. (Case #15) had a venous prothrombin time of 29% and a simultaneous capillary prothrombin time of 66%. Many sources of error are possible; excess of tissue fluids mixed with the capillary blood being among the foremost.

If the capillary prothrombin determination is to be of value even as a screening test for clotting disorders, several things must be considered: first, low prothrombin time by test (venous or otherwise) does not necessarily constitute hypoprothrombinemia, since in fibrinogen deficiency, or in the presence of coagulation-inhibiting or accelerating substances several variables may alter the so-called "prothrombin time[5, 6]." Second, there has been postulated a qualitative difference between infant and adult prothrombin[7, 8]. While this has not been substantiated, there is definite evidence that vitamin K, while it raises the prothrombin time values in newborns, has no effect on the incidence of neonatal hemorrhage[9, 10]. Third, there may be factors operating in infancy which allow certain individuals a better utilization of the small amounts of prothrombin present; for example, changes in prothrombin convertibility.

The need for further studies on prothrombin in infancy and childhood is great. A reliable capillary test would be of considerable practical value in the study of clotting disorders as it is carried out in many hospital routine "bleeding and clotting" workups. While the above described test is open to criticism, it appears to compare in accuracy with the venous one-stage prothrombin time determination as performed in routine hospital work. It is no more a true determination of prothrombin content of blood than the Quick method. If perfected, it might well perform useful service as standard adjunct to the grossly inadequate capillary "clotting time" test ordinarily used in hospital routines.

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TYPHOID MENINGITIS WITH SUBDURAL EFFUSION

A Case Report

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Pasquale W. Santagata*

J. S., a one year old colored male was admitted to Children's Hospital on June 6, 1954 with the chief complaint of fever and a cold of six days duration. The child was apparently well until six days before admission when he developed a cough, rhinitis and fever. The day following the onset of his illness, he was seen by a private physi-

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cian who began penicillin therapy and prescribed aspirin for fever recorded at 105°F. (rectal). That evening, since the child showed no improvement, he was again seen by his private physician who prescribed terramycin. Three days before hospitalization the child's temperature dropped and he seemed much improved. Two days before admission his temperature rose to 103° and remained in that vicinity up to the time of hospitalization. The mother stated that he had a mild diarrhea the first two days of his illness, averaging four bowel movements per day. Loose stools persisted until admission, but the infant had only one or two stools a day from the third day of his illness.

The patient had been in relatively good health before his present illness. He was born at 8 months gestation and weighed 5 lbs. 4 oz. During his first year of life he had received no immunizations. Development was normal and the family history was essentially non-contributory.

On admission, the child appeared extremely irritable and very dehydrated, his mucous membranes were very dry, there were thrush like spots in his mouth. There were no petechiae visible. His ears were slightly injected. The abdomen was distended and hyperresonant, his bowel sounds were high pitched. The liver edge was palpable 3 cm. below the left costal margin. The child had hyperactive deep tendon reflexes and 4 plus nuchal rigidity. The initial impression was pyogenic meningitis.

Spinal fluid findings on admission revealed clear fluid, protein 160 mgm. per cent, sugar 67 mgm. per cent, 106 white blood cells with 10 per cent polys and 90 per cent lymphocytes. Culture was positive for *Salmonella typhosa* three days after admission. Stool cultures were negative. The initial blood culture was positive for *Salmonella typhosa* and the organism was found to be very sensitive to all broad spectrum antibiotics.

Blood CO_2 on admission was 25 volumes per cent. Complete blood count revealed a hemoglobin of 6.5 grams, hematocrit of 21 per cent, 5,400 white blood cells with a differential of 52 per cent segs, 46 per cent lymphocytes and 2 per cent eosinophils. The total eosinophil count was 13/mm³. Urinalysis revealed four to five white blood cells/HPF. On the second hospital day a few rose spots were noticed on the child's abdomen. Febrile agglutination studies reported two days after admission showed a 1:320 typhoid "O" titer and a 1:320 typhoid "H" titer. Tube agglutination titers were 1:2560 for typhoid "H" and 1:320 for typhoid "O". Paratyphoid A and B agglutinations were negative.

The Maryland State Health Department traced the source of the child's infection

TABLE I

Subdural Effusion

Date	Amount	Appearance	Protein	Sugar	WBC	Polys	Lymphs
	cc.		*gm.*	*mg. %*			
6/11	15	Bloody	1.4	73	96	17	86
6/12	15	Bloody	2.1	60	115	9	91
6/14	20	Bloody	0.4	79	220	1	99
6/16	20	Bloody	0.69	73	147	2	92
6/18	25	Bloody	1.48	90	335	3	97
6/21	7	Bloody					
6/24	25	Bloody	3.20	63	139		
6/30	15	Bloody	2.94	83	2,500	2	98
Total	142						

back to a "Typhoid Mary", an elderly colored female named Mary, who had initiated eight cases of typhoid fever in the past four years. This woman had fed the child on a number of occasions.

Initially the patient received 160 mg. chloromycetin every six hours intravenously and 80 mg. of aureomycin every six hours intravenously. Intravenous solutions were used for his acidosis and dehydration. Sodium bicarbonate swabs were used to relieve the thrush like condition of his mouth. The child's hemoglobin and hematocrit eventually elevated to 10.8 gms. and 37 per cent respectively with transfusions of packed red blood cells.

On the fourth hospital day the dose of chloromycetin was increased to 300 mg. every six hours. On the fifth hospital day intravenous solutions were discontinued, chloromycetin 300 mg. was given orally every six hours.

On June 11th, the seventh day of hospitalization, a subdural tap was done because of seemingly poor clinical response. Under greatly increased pressure, 10 to 12 cc. of a blood tinged xanthrochromic fluid was obtained from the right side, 2 to 3 cc. of a similar appearing fluid was obtained from the left side. After the tap, the child's clinical condition improved dramatically. He became afebrile, he began to sit up and his appetite improved considerably. Subdural taps were repeated every other day. A total of 125 cc. of blood tinged xanthrochromic fluid was obtained from these taps. (See Table I).

The child was followed closely with hematocrits, hemoglobins, white blood cell and differential counts, spinal fluid examinations and total eosinophils. Repeated blood cultures were negative. Urine and stool cultures were never positive. Blood febrile agglutinations after 15 days of hospitalization were: typhoid "O" 1:320 plus, Paratyphoid A 1:320 plus, Paratyphoid B 1:80. Negative agglutinations were reported on spinal fluid, 15 days after admission but subdural fluid agglutinations were positive Typhoid "O" 1:80, typhoid "H" 1:160, and paratyphoid negative. Subdural fluid cultures were never positive.

EEG tracings four days following the first subdural tap showed low voltage; fast activity dominated the record. No amplitude asymmetry, phase reversal or evidence of focus were found. A second EEG report nine days later showed infrequent and high voltage, sharp biphasic waves appearing at random and in single synchronous bursts. Again no amplitude asymmetry or evidence of focus were noted.

Thirty days after admission craniotomy was performed through a bone flap on the left. About 30 cc. of subdural fluid was evacuated and a thick well-organized left subdural membrane was removed. The infant has done well post-operatively and it is anticipated that another craniotomy for removal of the subdural membrane on the right will be done.

DISCUSSION

Henderson[1] in 1948 described three cases of salmonella meningitis and collected 144 cases in the literature. At that time he did not include *Salmonella thyphosa* in his summary, but the typhoid bacillus is now recognized as a member of the genus salmonella. Beene[2] et al. were the first to include typhoid bacillus in a review of salmonella meningitis. Beene was able to collect 18 papers which referred to typhoid meningitis, following this Chigger[3] reported a case of typhoid meningitis treated with chloro mycetin with subsequent recovery. Until this case report no mention ha

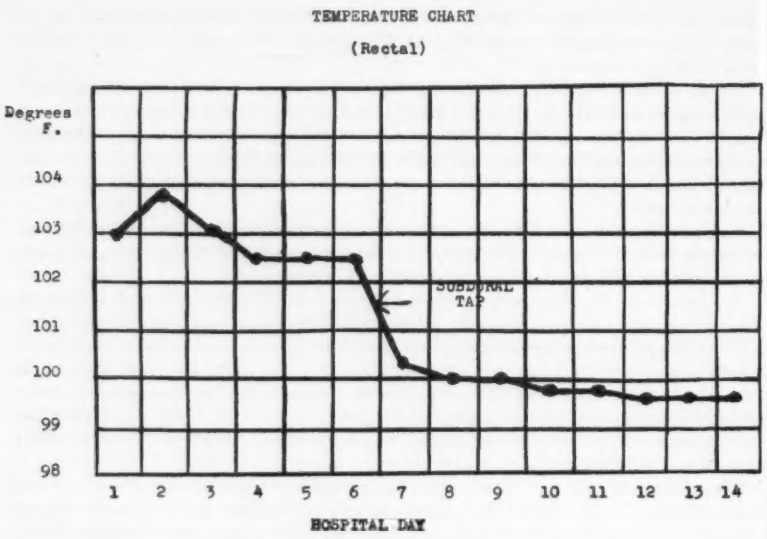

FIG. 1. Typhoid meningitis with subdural effusion

been made of complicating subdural effusion. We are certain the subdural tap performed on the seventh day of hospitalization initiated the drop in temperature and the good clinical response (see Fig. 1).

Penfield[4] first used the term subdural effusion in 1923 in his clinical presentation of a child with subdural effusion secondary to intracranial infection. He[5] further publicized the subject with his report on a series of experiments carried out on dogs to evaluate the amount of subdural fluid in normal animals. But serous subdural exudation was mentioned years before in 1916 by Schwartz[6] in his discussion on pachymeningitis. McKay, Morisette, Ingraham and Matson[7] in 1950 made reference to collections of subdural fluid in patients convalescing from influenzal meningitis. This has been followed by a number of reports indicating the frequency of complicating subdural effusions[8, 9, 10, 11, 12]. Margaret Smith et al.[13] found subdural effusions in 20 of 43 patients suffering with meningitis. The most common offending organisms were *H. Influenzae* and *D. pneumoniae*. Until the paper of Stevens and Williams[14], all reports in the literature have implicated meningitis as the cause of subdural effusion. They have suggested that subdural effusion may be more non-specific, being related to acute infections, severe chronic diarrhea, dehydration and inanition.

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PEPTIC ULCERS IN CHILDHOOD: A CASE REPORT OF A GASTRIC ULCER OCCURRING IN AN INFANT

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CASE REPORT

This colored male child was first admitted at the age of four months with the chief complaint of vomiting. The mother described this as "spitting up" which had started the first day the baby came home from the hospital and progressed to vomiting. The vomiting was non-projectile, contained previously ingested food but no blood or bile, and occurred at any time during the day or night. Bowel movements were normal during this period. He had also suffered from frequent colds and a cough, with noisy respirations, since birth. At four months of age there was an acute episode of respiratory distress, wheezing and cyanosis, for which conditions he was admitted to

another hospital for one week's duration. He was discharged without diagnosis, showing but little improvement.

The past history revealed the following facts: Pregnancy was complicated by ankle edema during the seventh month and headache and vomiting throughout the entire nine months. The mother admitted that the idea of another child was unwelcome. Delivery was at term and uncomplicated, and spontaneously. One sibling had eczema, and two had "spat up" briefly during the neo-natal period.

This child was breast fed from birth until four months of age when he began to receive evaporated milk with egg yolk, which seemed to reduce the vomiting temporarily. Vitamin supplements were adequate at all times.

Physical examination revealed a poorly developed, under-nourished, dehydrated, colored male weighing ten pounds. There was a generalized shotty lymphadenopathy. The head was symmetrical, and the anterior fontanelle, admitting two fingers, was depressed. There were no abnormalities found pertaining to the cardio-vascular, respiratory, gastro-intestinal, uro-genital, or central nervous sytems.

The impression at this time was that of malnutrition on account of vomiting, the cause of which was undetermined.

Laboratory investigations were numerous. They may be summarized as follows:

1. Urinalyses—Normal at monthly intervals.
2. Hemogram—Normal on admission, but developed an anemia (hypochromic, microcytic) for which he was transfused.
3. Blood sickling—Negative.
4. Serology—Negative.
5. Occult blood—(Guiac test) positive in stools and vomitus.
6. Stool trypsin—Positive (1:10); Fats and starches normal.
7. X-rays: chest—increased bronchovascular markings; long bones—negative; G.I. series—no diaphragmatic hernia. Incompetence of cardiac valve demonstrated.
8. Subdural punctures—Negative.
9. Bronchoscopy—Normal.
10. Esophagoscopy—Showed ectopic gastric mucosa protruding through the cardia of the stomach.

Various treatments were tried during this first hospitalization, which lasted from September 13, 1950 to March 2, 1951. Various types of milk were used in the formula, including olac and lactic acid milk. The child was fed by gavage for a time, and at other times the formula was thickened with cereal. Extra vitamin supplements, pancreatic granules, and anti-spasmotic drugs were tried without success. Hydration was maintained by clyses. Antibiotics were given for secondary infections.

Vomiting persisted throughout the hospital stay, but was slightly less during the last month when "propping" in the sitting position was instituted on around the clock basis. It was felt that there might be an emotional factor causing the vomiting, aided by the existence of an organic achalasis. The discharge diagnosis was that of achalasia and rumination.

At the age of seventeen months this child was admitted to the Harriet Lane Home where he stayed from August 15, 1951 until October 14, 1951, on account of vomiting and a failure to gain weight since two weeks of age.

It appeared that there was some retardation although the first tooth erupted at six months. He had not sat up until eleven months, and at seventeen months could neither crawl nor talk.

Physical examination revealed a child weighing 11.3 pounds (normal for age—

22 pounds). He was thin, malnourished, and underdeveloped with practically no subcutaneous fat. The child was irritable and uncooperative. He ate with a good appetite but vomited quite forcefully at once.

Laboratory work at this time showed:

1. CBC, urinalyses, and blood chemistries to be within normal limits.
2. Duodenal enzymes normal.
3. Barium swallow showed a pooling of barium proximal to, and a narrowing of the esophagus near the cardia of the stomach.

Vomiting continued until daily bouginage of the esophagus was instituted and a no. 30 bougie could be passed. The hemoglobin dropped to 9.0 gms. and a blood transfusion was given. He was discharged much improved.

This child was admitted to Children's Hospital for the second time on November 22, 1951, when he stayed for four days, the purpose of which was the performance of esophageal dilation.

This boy was now two years old and appeared to be physically and mentally retarded. He weighed 12.2 pounds. The possibility of a cerebral dysgenesis or mild amyotonia congenita was entertained.

The third admission lasted from January 15, 1952 until March 25, 1952. The symptoms were unimproved and the weight was only 13 pounds, 10 ounces.

On January 26, 1952 an exploratory laparotomy was performed, and a diaphragmatic hiatus was found to admit two fingers with ease.

On February 18, 1952 the diaphragmatic deficiency was repaired by means of a left trans-thoracic approach. The part of the stomach which was in the chest was freed from surrounding structures. The esophagus was brought down and the diaphragm sutured behind it.

The post-operative course was extremely stormy. The clinical picture was that of a high intestinal obstruction complicated by bronchopneumonia. On February 29, 1952 a third operation was performed through a right median incision revealing eighteen inches of greatly distended small bowel. Innumerable adhesions were released. Recovery was slow but uneventful.

The fourth admission occurred on July 24, 1952 and lasted until August 7, 1952, because of emeses of coffee ground material for two days with resultant dehydration and moribundity. The idea of a sub-clinical adrenal insufficiency was entertained. He was treated with I.V. fluids, blood transfusions and adrenal cortical extract.

At the time of the fifth admission (February 26, 1953–April 18, 1953) this child was three years old and weighed twenty-two pounds. The cause for admission again was uncontrollable vomiting. Examination revealed no positive findings. An esophagoscopy showed the presence of scar tissue near the cardia of the stomach but no stricture.

Sedative and anti-spasmodic drugs were used, and after six weeks he was finally placed on a regular diet. A psychogenic etiologic component was suspected as the vomiting seemed to be used to acquire attention. He received therapy from the psychiatry department, and vomiting decreased from twelve to fifteen to three to four times a day. The diagnosis of a non-demonstrable recurrent diaphragmatic hernia remained.

The sixth hospital admission, lasting from May 12, 1953 until May 16, 1953, was for treatment of a bronchopneumonia which responded to terramycin therapy. There had been occasional blood seen in the stools and vomitus since the previous admission.

The seventh admission was on account of abdominal pain and hematemesis. There were no abnormal physical findings. The hemoglobin was 6.8 gms., and two blood transfusions were given.

It was thought at this time that a gastric ulcer might be present, and a further admission for barium swallow, esophagoscopy, and eventual gastrotomy with excision of the ulcer was recommended. After four days of hospitalization he was discharged, not vomiting and weighing 24.4 pounds.

The eighth hospital admission occurred on October 26, 1953, at which time the patient was three and a half years old and weighed 24.0 pounds. He had vomited blood three days prior to this admission but had continued to eat well and had had no tarry stools or other complaints.

Examination revealed a small, pale child whose general condition was good. Two abdominal scars and one thoracic scar were noted. The liver edge was palpable below the right costal margin.

The hemoglobin was 5.8 gms. with a hematocrit of 23%. Two blood transfusions were given, raising the hemoglobin to 11.8 gms.

Esophagoscopy revealed a normal esophagus with a small bleeding area just proximal to the gastric side of the cardia.

Barium series showed no evidence of a diaphragmatic hernia or gastric ulcer.

On November 11, 1953 another laparotomy was performed. Through this incision the stomach was incised showing an ulcer at the cardia which was technically impossible to remove by the abdominal approach. Biopsy was taken from the ulcer, and a sub-total gastrectomy was performed.

Routine post-operative care was instituted. Intravenous fluids and continuous Wangensteen suction were continued until borborygmus was heard on the third post-operative day. Infection was controlled by penicillin-streptomycin therapy for five days. Two small blood transfusions were given as indicated. Serum electrolytes were estimated daily for the three post-operative days, and at no time was there any discrepancy from the normal.

The post-operative course was smooth and uneventful. He was able to take a soft diet on the eighth day after surgery. He was discharged two weeks after the operation having gained a pound in weight and was symptom free. Follow up in the out-patient clinic has shown a steady general improvement with no recurrence of symptoms.

Pathology Report

1. Biopsy of ulcerated area in cardia of stomach near esophagus showed only a moderate submucosal inflammatory reaction with an inflammation of the mucosa.

2. Stomach—Gross specimen consists of a portion of stomach in the region of the greater curvature 7.5 x 3.5 cm. The mucosa is a pink to a dark red, and the rugae are not prominent. Microscopic—A small bleeding point was seen between the glandular structures and the muscularis.

DISCUSSION

Mary H. McConnell, M.D.

The incidence of peptic ulceration is variously reported from 0.1 to 2.32 per cent, with equal sex distribution in early life[1]. The preponderance of duodenal over gastric ulcer is shown in numerous publications in ratios varying from 2:1 to 6:1. Although chronic peptic ulcer is rarely reported before puberty the history of ulcer in adult life often dates back to childhood when the condition existed unrecognized[2, 3].

Most cases present no obvious cause and no significant cerebral lesions have been observed in infants. Gastric acidity deserves special attention. During the first year of life gastric acidity shows a rapid increase in concentration with a probable increase in volume. At the end of this period the reaction of the stomach to a test meal of milk closely resembles that of an adult, for the maximum and average values are almost identical in the two age groups[4]. High gastric acidity could appear per se to be an important factor in the production of peptic ulceration at an early age. Circulatory instability, especially in prolonged labor may cause devitalization of duodenal mucosa and a damaged area liable to digestion by gastric juice.

Trauma may damage the gastrointestinal mucosa, directly by instruments introduced for clearing the trachea which may enter the stomach and abrade the mucosa, or indirectly in association with pyloric stenosis by hyperperistalsis and vomiting.

Other etiologic factors are sepsis, burns, and scalds, during the course of the various exanthemata and toxemic conditions. With marasmus in athreptic infants (and in prematures) the gastrointestinal mucosa tends to be thin and atrophic and less resistant to the digestive action of gastric juice[1].

Duodenal ulcers usually occur above the ampulla of Vater or on the posterior wall. Two-thirds are single ulcers. The diameter varies up to 1.5 cm. Histologically there is an absence of any inflammatory reaction in acute ulcers, the lesions being purely destructive[1].

Ulceration has been reported to occur in utero. In the newborn the course is precipitous and hemorrhage is the most important and characteristic sign. Perforation may occur. The lesion is acute without cellular reaction or bacterial invasion. The ratio of duodenal to gastric ulcer is 2 to 1[5]. The prognosis is poor owing to the frequency and severity of hemorrhage.

During the first 24 months the incidence is less frequent, the clinical picture is obscure and hemorrhage or perforation occur in the great majority. Symptoms include refusal of feedings, anemia, vomiting, diarrhea, melena, failure to gain weight satisfactorily and gradual marasmus. There may be a persistant pylorospasm. The lesion has a sub-acute or chronic base with many bacteria. The ratio of duodenal and pyloric to gastric ulcer is 5:1. Symptoms may recede and health be regained[5].

From 2 to 6 years ulcerations are few in number. Chronicity, hemorrhage, perforation and stenosis are outstanding features. In patients over 6 years the symptom complex may be as clear as in adults, but more frequently is atypical with accentuation of the symptoms of pyloric stenosis and perforation[5]. Typical night or pre-breakfast pain is uncommon in the younger patients but may occur in older age groups. This is likewise true of symptomatic relief obtained by ingestion of food or alkali. Acid eructations or pyro-

sis are not noted. Abdominal tenderness is more frequently epigastric in older groups, periumbilical in younger patients[6].

The diagnosis is based primarily on roentgenographic examination. Gastric analyses are not very practical and results are not so significant[2].

Complications are hemorrhage, pyloric obstruction and perforation, the latter producing a peritonitis which is generally afebrile since duodenal contents are normally sterile[1].

The treatment of uncomplicated cases is medical with special diets and administration of alkalis and antispasmotics. The indications for surgery are perforation, obstruction, recurrent or uncontrollable bleeding and intractable pain not responsive to medical treatment[2].

SUMMARY

An interesting case report of a gastric ulcer occurring in an infant and successfully treated by subtotal gastrectomy has been reported. A short discussion of ulcers in childhood with emphasis on etiology, symptomatology, and diagnosis is included.

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ORBITAL CELLULITIS IN CHILDREN

A Special Report

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Although orbital cellulitis and the conditions which incite it have been greatly curtailed and modified by antibiotics, it still occurs relatively frequently and is potentially dangerous enough to be of interest to the pediatrician unless adequately treated. The potential dangers of orbital cellulitis are twofold, there being a possibility of loss of vision, and the more remote but still possible threat to life itself. The danger of loss of vision develops from several possible sources, including (a) proptosis with resulting exposure keratitis; (b) stretching of the optic nerve with loss of func-

tion; (c) thrombophlebitis of the orbital blood vessels; (d) compression of the central retinal artery with optic exudation and congestion. The danger to life arises from: (a) the profound septic state which develops from absorption of massive amounts of toxins; and, (b) the ease of extension of the infection from the orbit to the cranial cavity causing meningitis, subdural or cerebral abscess, or cavernous sinus thrombosis, any one of which can be fatal.

Anatomically the orbit presents several interesting considerations which enter into the development of orbital cellulitis and must be kept in mind in the treatment of the condition. The orbit itself contains no lymphatic system so that the protective mechanisms are limited to the confining qualities of the fascial sheaths, local phagocytic elements, and the digestive action of the orbital fat. Two thirds of the bony walls of the orbit are thin plates which delimit sinuses and these are often incomplete and in some areas are composed of mucous membrane only. In the orbit there are many venous connections to wide areas of drainage, especially through the intimately connected venous plexuses of the paranasal sinuses, turbinates, and the cavernous sinus. Finally, the fascial sheaths of the orbit form a confined area so that the increased pressure following inflammation necessarily seeks release anteriorly, pushing the orbital contents ahead of it; the increased pressure in the tissue augments the virulence of the organisms and tends to cause early necrosis.

Although there are many exciting causes, the etiology of orbital cellulitis in the vast majority of cases is an extension from neighboring structures, usually the paranasal sinuses. The most probable estimate is that about 70 per cent of cases follow paranasal sinusitis, which itself frequently follows acute infections such as scarlet fever, measles, diphtheria, and influenza in children. Due to the age at which the sinuses develop, the ethmoids, which are present at birth are the most common offenders in infants and young children, with the maxillaries a close second; after the age of 9 or 10 years the frontals, which are now developing, become important; in adult life all three may be equally important. The extension from the paranasal sinuses to the orbit can be a direct penetration through the orbital walls when they are thin or defective; extension may be by the veins as a thrombophlebitis which may cause a cellulitis directly, or secondarily by a subperiorbital infection and consequent spread to the orbital fat; the spread may be indirect by metastasis of the organisms through the blood stream.

Dental infection may be an important exciting cause of orbital cellulitis. It may be spread by direct extension of a subperiosteal abscess over the anterior surface of the maxilla to the subperiorbita and thence directly to the orbital contents, or it may extend as thrombophlebitis via the pterygoid plexus to the orbital veins. The anatomical relationship of the first upper

molar to the antrum makes it quite important in dental infections. Prior to antibiotic therapy several cases of blindness and some of death were reported following extraction of abscessed first upper molars. In infants the permanent tooth buds lie just beneath the immature antrum and are quite close to the floor of the orbit. An infection, usually staphylococcal, of the upper deciduous unerupted first molar in infants is followed by a maxillary osteomyelitis which is easily spread through the antrum and into the orbit.

Face and lid infections lead to orbital cellulitis quite frequently. In the case of erysipelas the course was by direct extension through lid involvement, while in the case of focal infections such as furuncles, hordeola, and acne lesions, the course was by venous extension. Rarely, deeper infections of the facial structures such as parotitis and lymphadenitis can be the exciting factor.

Ear infections may uncommonly lead to orbital cellulitis either by causing an intracranial sinus (cavernous) thrombosis which in turn infects one or both orbits by direct extension, or by direct metastatic infection or extension of thrombophlebitis through the venous plexuses of the petrotympanic fissure, sphenopalatine fossa, and inferior orbital fissure. Intracranial infections can obviously be either a cause or result of orbital cellulitis. Extradural or cerebral abscesses can directly erode the orbital walls while cavernous sinus thrombosis may extend through venous connections as a thrombophlebitic process to the orbital veins. Intraorbital and intraocular infections are rarely the primary site.

Constitutional diseases such as typhoid, rheumatism, tuberculosis and syphilis can give inflammatory lesions in the orbit varying from low grade or subacute cellulitis to an acute purulent cellulitis. Severe acute orbital disease has been seen in tularemia, coccidiosis, tick fever, and other rickettsial and viral diseases. These are usually a manifestation of the systemic disease and not a result of extension or penetration. In some cases the low grade inflammation forms a mass in the orbit, the so-called "pseudo-tumor".

Ocular signs and symptoms of orbital cellulitis include: (a) proptosis, which may or may not be axial, depending on whether the process has localized or is diffuse in the orbit; it is irreducible and can rarely be extreme to the point of luxation of the globe; (b) edema of the lids which may become brawny; it is usually accompanied by redness from dilated vessels; a passive reactive edema characteristically occurs in the lids of the other eye and over the infected sinuses; (c) chemosis which may become severe enough that the conjunctiva protrudes, becomes desiccated, and finally necrotic; a passive chemosis similar to and accompanying the passive lid edema may occur in the other eye; (d) external ophthalmoplegia which may vary from a partial and mild form to a total ophthalmoplegia; (e)

pain; (f) visual loss varying from transient field constriction to total loss of field and even permanent blindness in some complicated cases.

General signs and symptoms are those of any severe toxic state, namely: (a) fever; (b) nausea; (c) vomiting; (d) prostration; (e) delirium, coma, and/or convulsions occasionally can develop and are often due partly to the cellulitis per se and partly to the exciting cause of the cellulitis; (f) slow pulse which is often found and is thought to be due to the vagal stimulation through the oculo-cardiac reflex.

Orbital cellulitis tends to be rapid in onset, fulminating by nature, and violent in its complicating developments. When recovery does occur, it does so relatively slowly. Talkovskee, in 1940, found that from 93 cases, 17 died; 6 from cavernous sinus thrombosis, 3 from meningitis, 3 from cerebral abscess, 3 from septicemia, 1 from pneumonia, and 1 from empyema. Again it must be remembered that the course of the disease is now radically altered by the use of antibiotics.

Complications before the advent of antibiotics, and to a certain extent today, are the rule rather than the exception. They are numerous and include: (a) formation of orbital abscess; (b) keratitis with possible perforation and visual loss both from exposure following keratitis and from interference with the nerve supply of the cornea; (c) optic nerve atrophy which may follow traction, mechanical pressure, compression of the central retinal vessels, or direct invasion of the optic nerve by the infective process; the incidence was 20 per cent before antibiotics; (d) retinal hemorrhages which occasionally follow thrombosis or embolism; sometimes there is a septic uveitis with subsequent retinal detachment and rarely panophthalmitis; (e) temporal or parotid abscess occasionally develops by spreading phlebitis through the maxillary and pterygoid plexuses; (f) cavernous sinus thrombosis and/or meningitis by posterior venous spread of the phlebitis or by direct extension through the optic canal or the orbital fissures; (g) development of general septicemia. Prior to the use of antibiotics 20 per cent of the non-fatal cases were left blind in the affected eye and an additional 13 per cent had grave visual loss. Occasional cases of blindness still occur even with the use of antibiotics.

The signs and symptoms which may indicate the onset of serious complication include: (a) engorgement of the forehead veins; (b) development of a rapid pulse; (c) development of total ophthalmoplegia in one eye and then the other; (d) development of intense headache, vomiting, and severe prostration.

It is obvious from the preceding material that the mainstay in the therapy of orbital cellulitis is the use of antibiotics. Since the complications of this condition can be rapid and fatal, it is probably foolhardy to withold antibiotics until the organism is identified. As soon as smear and culture

samples from the conjunctiva and any other suspected primary etiological site have been obtained, heavy, "blunderbuss" doses of antibiotic combinations are warranted. The combinations should be such that the bacterial spectrum is adequately covered. If and when the organism is identified, and the sensitivity tests returned, the antibiotics may be changed to conform to the findings of the laboratory.

Following the institution of antibiotic therapy an immediate search for the primary etiological site and treatment of the primary condition should be started. In sinusitis it is essential to establish and maintain drainage with nose drops and nasal irrigation. Later surgical correction of such conditions as dental infections and mastoiditis must wait for control of the infective process.

General supportive therapy, including maintenance of electrolyte balance, nutrition, and sedation is basic. Surgical incision of the orbit through the lids may have to be done when and if an abscess develops. Abscesses can usually be distinguished by indurated areas which fail to subside under intensive therapy. In general, it is bad practice to incise unless localization is present. If it is reasonably certain that orbital abscess has developed but no site of localization can be found, incision can be best done through the upper lid at the middle of the upper orbital rim, extending back into the middle zone of the eye. In this position there is little danger of damage to the nerves and blood vessels of the orbit. Incision through the cul de sac, especially around the inner canthus should be avoided.

In cases where the process is fulminating and loss of vision is occurring rather rapidly, indicating a severe septic process under pressure, surgical incision is imperative. If no suppuration is encountered in this incision, and the tension appears to be severe to the point of strangulation, incision of the lateral tarsal ligament and the external rectus may be necessary to allow access to the central fascial compartment. In rare cases before antibiotics the eye which had already lost its function had to be enucleated to establish drainage.

Case 1

This is a fairly typical uncomplicated case of orbital cellulitis following ethmoiditis, which in turn followed an acute febrile disease.

A. W., a 5 year old male, was admitted to Childrens Hospital on March 11, 1954 with a history of having measles and an accompanying conjunctivitis three weeks previously. The measles and conjunctivitis cleared well, but two days prior to admission the patient rather suddenly developed swelling, pain and loss of motion of the left eye. One day prior to admission the patient was seen in the out patient clinic with the above findings and a temperature of 105 degrees (rectal). On the day of admission the patient presented himself with a temperature of 102 degrees and a general increase in the severity of the symptoms. Positive physical findings included: (a) mucopurulent rhinorrhea; (b) moderate pharyngitis; (c) gross, brawny edema

of the lids and periorbital structures of the left eye; (d) edema across the bridge of the nose and a passive reactive edema of the right eye; (e) severe chemosis; (f) proptosis of the left eye, slightly downward and outward; (g) partial ophthalmoplegia of the left eye with motion only in a restricted field up and nasally.

Conjunctival smears and cultures were negative. Blood cultures were negative. Ear, nose, and throat survey and x-rays revealed a pan-sinusitis involving all sinuses but the frontals which were undeveloped. Treatment was immediately initiated and consisted of terramycin suspension 125 mgm. q. 4 h., penicillin, 400,000 units b.i.d., cold compresses to the left eye t.i.d., neosynephrine nose drops q. 3 h., nasal irrigation with warm saline daily, and atropine, ½%, gtts. i in the left eye, b.i.d.

Over a period of four days the temperature dropped to normal; the chemosis, edema, and proptosis disappeared gradually over a period of eleven days at which time the extraocular motion had returned to full range. Visual loss was not found. The patient is being followed in the out-patient department.

Case 2

This case illustrates the complications of orbital abscess and meningitis which may follow orbital cellulitis.

B. R., a 23 month old white female, was admitted to Childrens Hospital on September 15, 1948 with a history of rapid swelling of the left eye three days previously. On admission she showed: (a) pharyngeal injection; (b) temperature of 105 degrees; (c) marked lid edema of the left eye; (d) severe chemosis of the left eye; (e) pain. She was diagnosed as orbital cellulitis and treatment was instituted, consisting of penicillin, 50,000 units q. 2 h., hot compresses to the eye q. 2 h., and clyses as needed. Sinus examination and x-ray revealed ethmoiditis. Conjunctival culture showed hemolytic *Staphylococcus aureus.*

On the second hospital day the patient became lethargic and finally comatose. A diagnosis of meningitis and possible cavernous sinus thrombosis was made. Lumbar puncture revealed cloudy fluid with a cell count of 450, of which 66 per cent were polys. 25,000 units of streptomycin and 5000 units of penicillin were injected into the spinal canal and the systemic medication increased to: streptomycin, 125,000 units q. 3 h.; penicillin, 50,000 units q. 2 h.; and sulfadiazine, 1 gram q. 8 h. Repeat spinal tap on the following day revealed 557 cells in cloudy fluid and a smear showed Gram positive diplococci and some Gram negative cocci present.

On September 24, after the grave condition had improved sufficiently, incision and drainage of the left orbit was carried out through the inner one third of the upper lid with the release of about 100 cc. of frank pus which on culture revealed *Staphylococcus aureus.*

Slow recovery followed with the temperature finally reaching normal on September 28, 1948. The patient was discharged to be followed in the out patient clinic on October 14, 1948.

Case 3

This case illustrated orbital cellulitis in which dental infection was found to play the primary role.

G. T., a nine year old male, was admitted to Childrens Hospital on November 11, 1946 with a history of swelling of his right eye and a dental infection for the previous week. The swelling had been much more severe in the two days preceding admission and by the date of admission had swollen to the point of complete closure of the lids. This was accompanied by persistent vomiting and fever.

On admission the patient had a grossly inflamed and swollen right eye. The swelling was fluctuant and extended laterally over the zygoma to the preauricular area. The temperature was 101 degrees. Ear, nose and throat consultation and x-rays failed to reveal sinus pathology.

Treatment consisted of penicillin, 30,000 units stat, and 10,000 units q. 3 h.; magnesium sulfate soaks to the eye q.i.d., and sulfadiazine, 1 gram t.i.d. Incision and drainage was carried out with the release of pus. Dental examination revealed infection of the upper deciduous molars and it was recommended that extraction of both upper and lower molars be done when the condition warranted.

The patient responded well and on November 18, 1946 was discharged to be followed in dental clinic for the definitive therapy recommended.

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